WEST Search History

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DATE: Thursday, June 23, 2005

Hide?	Set Name	Query	Hit Count
	DB=PGPB,USPT,EPAB,JPAB,DWPI; THES=ASSIGNEE; PLUR=YES; OP=ADJ		
☐	L7	stegmann T.in.	5
	DB=USPT; THES=ASSIGNEE; PLUR=YES; OP=ADJ		
☐	L6	L5	5
	DB=PGPB,USPT,EPAB,JPAB,DWPI; THES=ASSIGNEE; PLUR=YES; OP=ADJ		
☐	L5	L4 and human	22
☐	L4	L3 and coronary	23
☐	L3	L2 and FGF	27
☐	L2	L1 and ischemic	136
☐	L1	revascularizing	315

END OF SEARCH HISTORY

FILE COVERS 1907 - 23 Jun 2005 VOL 142 ISS 26
FILE LAST UPDATED: 22 Jun 2005 (20050622/ED)

New CAS Information Use Policies, enter HELP USAGETERMS for details.

This file contains CAS Registry Numbers for easy and accurate substance identification.

```
=> "vascular endothelial growth factor"
      142221 "VASCULAR"
          4 "VASCULARS"
      142224 "VASCULAR"
             ("VASCULAR" OR "VASCULARS")
       93569 "ENDOTHELIAL"
          10 "ENDOTHELIALS"
       93573 "ENDOTHELIAL"
             ("ENDOTHELIAL" OR "ENDOTHELIALS")
     1197732 "GROWTH"
        4197 "GROWTHS"
     1199890 "GROWTH"
             ("GROWTH" OR "GROWTHS")
      902352 "FACTOR"
      804002 "FACTORS"
     1424586 "FACTOR"
             ("FACTOR" OR "FACTORS")
L1      15357 "VASCULAR ENDOTHELIAL GROWTH FACTOR"
             ("VASCULAR"(W)"ENDOTHELIAL"(W)"GROWTH"(W)"FACTOR")

=> revascular iz5
L2    5881693 5

=> L1 and l2
L3       3740 L1 AND L2

=> "ischemic" and L3
       41674 "ISCHEMIC"
           7 "ISCHEMICS"
       41676 "ISCHEMIC"
             ("ISCHEMIC" OR "ISCHEMICS")
L4        221 "ISCHEMIC" AND L3

=> human and L4
     1441812 HUMAN
      327188 HUMANS
     1605232 HUMAN
```

```
          (HUMAN OR HUMANS)
L5        124 HUMAN AND L4

=> physical (w) glue
      410840 PHYSICAL
          20 PHYSICALS
      410860 PHYSICAL
             (PHYSICAL OR PHYSICALS)
      464811 PHYS
           9 PHYSES
      464820 PHYS
             (PHYS OR PHYSES)
      814088 PHYSICAL
             (PHYSICAL OR PHYS)
       15341 GLUE
        4605 GLUES
       17735 GLUE
             (GLUE OR GLUES)
L6          1 PHYSICAL (W) GLUE

=> physiological (w) glue
       47356 PHYSIOLOGICAL
      256155 PHYSIOL
           1 PHYSIOLS
      256155 PHYSIOL
             (PHYSIOL OR PHYSIOLS)
      282106 PHYSIOLOGICAL
             (PHYSIOLOGICAL OR PHYSIOL)
       15341 GLUE
        4605 GLUES
       17735 GLUE
             (GLUE OR GLUES)
L7          2 PHYSIOLOGICAL (W) GLUE

=> L7 and L5
L8          0 L7 AND L5

=> heparin and L5
       45329 HEPARIN
        1716 HEPARINS
       45437 HEPARIN
             (HEPARIN OR HEPARINS)
L9          6 HEPARIN AND L5

=> "coronary artery stenosis"
       58569 "CORONARY"
```

```
        225 "CORONARIES"
      58635 "CORONARY"
            ("CORONARY" OR "CORONARIES")
     118088 "ARTERY"
      32019 "ARTERIES"
     129534 "ARTERY"
            ("ARTERY" OR "ARTERIES")
       4910 "STENOSIS"
L10     374 "CORONARY ARTERY STENOSIS"
            ("CORONARY"(W)"ARTERY"(W)"STENOSIS")

=> L10 and L5
L11       0 L10 AND L5

=> L10 and L4
L12       0 L10 AND L4

=> D L9 IBIB ABS 1-6
=> "endothelia cell growth factor"
        1099 "ENDOTHELIA"
     1927276 "CELL"
     1699605 "CELLS"
     2565531 "CELL"
             ("CELL" OR "CELLS")
     1197732 "GROWTH"
        4197 "GROWTHS"
     1199890 "GROWTH"
             ("GROWTH" OR "GROWTHS")
      902352 "FACTOR"
      804002 "FACTORS"
     1424586 "FACTOR"
             ("FACTOR" OR "FACTORS")
L13       0 "ENDOTHELIA CELL GROWTH FACTOR"
            ("ENDOTHELIA"(W)"CELL"(W)"GROWTH"(W)"FACTOR")

=> "astroglial growth factor"
        2419 "ASTROGLIAL"
     1197732 "GROWTH"
        4197 "GROWTHS"
     1199890 "GROWTH"
             ("GROWTH" OR "GROWTHS")
      902352 "FACTOR"
      804002 "FACTORS"
     1424586 "FACTOR"
             ("FACTOR" OR "FACTORS")
L14      21 "ASTROGLIAL GROWTH FACTOR"
```

("ASTROGLIAL"(W)"GROWTH"(W)"FACTOR")

=> L14 and L2
L15 6 L14 AND L2

=> D L15 IBIB ABS 1-6

=> "retinal-derived growth factor"
27702 "RETINAL"
388 "RETINALS"
27735 "RETINAL"
("RETINAL" OR "RETINALS")
755252 "DERIVED"
2 "DERIVEDS"
755254 "DERIVED"
("DERIVED" OR "DERIVEDS")
1197732 "GROWTH"
4197 "GROWTHS"
1199890 "GROWTH"
("GROWTH" OR "GROWTHS")
902352 "FACTOR"
804002 "FACTORS"
1424586 "FACTOR"
("FACTOR" OR "FACTORS")
L16 4 "RETINAL-DERIVED GROWTH FACTOR"
("RETINAL"(W)"DERIVED"(W)"GROWTH"(W)"FACTOR")

=> ischemic and L16
41674 ISCHEMIC
7 ISCHEMICS
41676 ISCHEMIC
(ISCHEMIC OR ISCHEMICS)
L17 0 ISCHEMIC AND L16

=> L1 and ischemic
41674 ISCHEMIC
7 ISCHEMICS
41676 ISCHEMIC
(ISCHEMIC OR ISCHEMICS)
L18 883 L1 AND ISCHEMIC

=> "throractomy incision"
0 "THRORACTOMY"
3218 "INCISION"
834 "INCISIONS"
3797 "INCISION"

("INCISION" OR "INCISIONS")
L19 0 "THRORACTOMY INCISION"
("THRORACTOMY"(W)"INCISION")

=> coronary (w) artery (w) stenosis
58569 CORONARY
225 CORONARIES
58635 CORONARY
(CORONARY OR CORONARIES)
118088 ARTERY
32019 ARTERIES
129534 ARTERY
(ARTERY OR ARTERIES)
4910 STENOSIS
L20 374 CORONARY (W) ARTERY (W) STENOSIS

=> L20 and L18
L21 0 L20 AND L18

=> L20 and L18
L22 0 L20 AND L18

=> L20 and l1
L23 0 L20 AND L1

=> "heparin binding growth factor=> aFGF and L20
823 AFGF
4 AFGFS
823 AFGF
(AFGF OR AFGFS)
L24 0 AFGF AND L20

=> FGF and L20
11142 FGF
1023 FGFS
11273 FGF
(FGF OR FGFS)
L25 0 FGF AND L20

=> "eye-derived growth factor"
104518 "EYE"
23506 "EYES"
112942 "EYE"
("EYE" OR "EYES")
755252 "DERIVED"
2 "DERIVEDS"

```
      755254 "DERIVED"
             ("DERIVED" OR "DERIVEDS")
     1197732 "GROWTH"
        4197 "GROWTHS"
     1199890 "GROWTH"
             ("GROWTH" OR "GROWTHS")
      902352 "FACTOR"
      804002 "FACTORS"
     1424586 "FACTOR"
             ("FACTOR" OR "FACTORS")
L26       52 "EYE-DERIVED GROWTH FACTOR"
             ("EYE"(W)"DERIVED"(W)"GROWTH"(W)"FACTOR")

=> L26 and ischemic
       41674 ISCHEMIC
           7 ISCHEMICS
       41676 ISCHEMIC
             (ISCHEMIC OR ISCHEMICS)
L27        0 L26 AND ISCHEMIC

=> prostatropin and l2
           6 PROSTATROPIN
          24 PROSTATROPINS
          26 PROSTATROPIN
             (PROSTATROPIN OR PROSTATROPINS)
L28        9 PROSTATROPIN AND L2

=> D L28 IBIB ABS 1-9

=> glial maturation factor
       22883 GLIAL
           6 GLIALS
       22885 GLIAL
             (GLIAL OR GLIALS)
       65951 MATURATION
          77 MATURATIONS
       65982 MATURATION
             (MATURATION OR MATURATIONS)
      902352 FACTOR
      804002 FACTORS
     1424586 FACTOR
             (FACTOR OR FACTORS)
L29       23 GLIAL MATURATION FACTOR
             (GLIAL(W)MATURATION(W)FACTOR)

=> ishcemic and l29
```

2 ISHCEMIC
L30 0 ISHCEMIC AND L29

=> FGF
L1 20226 FGF

=> vascular iz4
L2 7099699 4

=> L1 and L2
L3 4515 L1 AND L2

=> ischemic and l3
L4 101 ISCHEMIC AND L3

=> neoagiogenesis
L5 1 NEOAGIOGENESIS

=> human and L3
L6 1745 HUMAN AND L3

=> L6 and L4
L7 43 L6 AND L4

=> acidic and l7
L8 3 ACIDIC AND L7

⇨ L7
⇨
L13 43 L7

=> intramyocardiaS3
L14 0 INTRAMYOCARDIAS3

=> myocardia2
SYSTEM LIMITS EXCEEDED - SEARCH ENDED
The search profile you entered was too complex or gave too many answers. Simplify or subdivide the query and try again. If you have exceeded the answer limit, enter DELETE HISTORY at an arrow prompt (=>) to remove all previous answers sets and begin at L1. Use the SAVE command to store any important profiles or answer sets before using DELETE HISTORY.

=> myocardiacally
L15 0 MYOCARDIACALLY

=> hear
L16 1969 HEAR

=> L13 and L16
L17 0 L13 AND L16

=> coronary and L13
L18 21 CORONARY AND L13

=> D L18 IBIB ABS 1-21

L9 ANSWER 1 OF 3 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1998:339214 CAPLUS
DOCUMENT NUMBER: 129:63235
TITLE: Intramyocardial infusion of **FGF**-1 mimics **ischemic** preconditioning in pig myocardium
AUTHOR(S): Htun, Patrik; Ito, Wulf D.; Hoefer, Imo E.; Schaper, Jutta; Schaper, Wolfgang
CORPORATE SOURCE: Max-Planck-Inst. Physiol. Clin. Res., Bad Nauheim, D-61231, Germany
SOURCE: Journal of Molecular and Cellular Cardiology (1998), 30(4), 867-877
CODEN: JMCDAY; ISSN: 0022-2828
PUBLISHER: Academic Press Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Previous studies on the mRNA and protein level suggested a cardioprotective role of **FGF**-1. These presumed actions of **FGF**-1 and **FGF**-2, as well as the underlying mechanisms, were investigated in this study. **Human** recombinant **FGF**-1 (0.5 μg/mL, 20 μl/min) and **FGF**-2 (2 μg/mL) were applied by direct intramyocardial infusion (IM) for 60 min prior to a 60 min LSD-occlusion and 120 min reperfusion. Myocardial infarction compared to the region at risk was significantly decreased by **FGF**-1 and **FGF**-2 treatment (**FGF**-1: 51.8%, **FGF**-2: 57.3% vs. control 83.4%). The increase in survival time was about 33 min. and equaled that of **ischemic** preconditioning. This effect was caused by the mitogenic part of the mol., since infusion of a truncated version of **FGF**-1 (0.5-1 μg/mL), lacking mitogenicity but maintaining hemodynamic activity, did not induce cardioprotection (78.3% vs. control 83.4%). Suramin (0.5 μg/mL) prevented the observed cardioprotection (77.0% vs. control 83.4%) proving that the cardioprotective effect is receptor-mediated. Genistein (0.5 μg/mL), an inhibitor of tyrosine kinases, abolished the cardioprotection as well (77.2% vs. control: 83.4%). Immunohistochem. staining revealed an uptake and translocation of exogenous **FGF**-1 to a (peri-)nuclear localization in myocytes and into non-myocytes for **FGF**-2. The authors conclude that both **FGF**-1 and **FGF**-2 are cardioprotective (**FGF**-1 being more active on a molar basis), and mimic **ischemic** preconditioning. Their actions are receptor-mediated and receptor activation is involved. Uptake and transport to a (peri-)nuclear localization, seems to be a pathway of minor relevance, since it could not be blocked by tyrosine kinase receptor localization, seems to be a pathway of minor relevance, since it could not be blocked by tyrosine kinase receptor inhibition. Tyrosine kinase-coupled receptor occupation in general is not protective as demonstrated by the lack of effect with VEGF-infusion.
REFERENCE COUNT: 30 THERE ARE 30 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 2 OF 3 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
ACCESSION NUMBER: 1998:275710 BIOSIS
DOCUMENT NUMBER: PREV199800275710
TITLE: Intramyocardial infusion of **FGF**-1 mimics **ischemic** preconditioning in pig myocardium.
AUTHOR(S): Htun, Patrik; Ito, Wulf D.; Hoefer, Imo E.; Schaper, Jutta; Schaper, Wolfgang [Reprint author]
CORPORATE SOURCE: Max-Planck-Inst. Physiological and Clinical Res., W.G. Kerckhoff-Inst., Dep. Experimental Cardiol., Benekestrasse 2, D-61231 Bad Nauheim, Germany
SOURCE: Journal of Molecular and Cellular Cardiology, (April, 1998) Vol. 30, No. 4, pp. 867-877. print.
CODEN: JMCDAY. ISSN: 0022-2828.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 24 Jun 1998
Last Updated on STN: 24 Jun 1998

AB Previous studies on the mRNA and protein level suggested a cardioprotective role of **FGF**-1. These presumed actions of **FGF**-1 and **FGF**-2, as well as the underlying mechanisms, were investigated in this study. **Human** recombinant **FGF** -1 (0.5 mug/ml, 20mul/min) and **FGF**-2 (2mug/ml) were applied by means of direct intramyocardial infusion (IM) for 60 min prior to a 60 min LAD-occlusion and 120 min reperfusion. Myocardial infarction compared to the region at risk was significantly decreased by **FGF**-1 and **FGF**-2 treatment (**FGF**-1: 51.8 +- 7.7%, respectively, **FGF**-2: 57.3 +- 6.5% v control 83.4 +- 2.8%, P < 0.05). The increase in survival time was about 33 min, and equalled that of **ischemic** preconditioning. This effect was caused by the mitogenic part of the molecule, since infusion of a truncated version of **FGF** -1 (0.5-1 mug/ml), lacking mitogenicity but maintaining hemodynamic activity, did not induce cardioprotection (78.3 +- 0.73% v control 83. 4 +- 2.8%). Suramin (0.5mug/ml) prevented the observed cardioprotection (77.0 +- 1.2% v control 83.4 +- 2.8%) proving that the cardioprotective effect is receptor-mediated. Genistein (0.5 mug/ml), an inhibitor of tyrosine kinases, abolished the cardioprotection as well (77.2 +- 2.4% v control: 83.4 +- 2.8%). Immunohistochemical staining revealed an uptake and translocation of exogenous **FGF**-1 to a (peri-)nuclear localization in myocytes and into non-myocytes for **FGF**-2. We conclude that both **FGF** -1 and **FGF**-2 are cardioprotective (**FGF**-1 being more active on a molar basis), and mimic **ischemic** preconditioning. Their actions are receptor-mediated and receptor activation is involved. Uptake and transport to a (peri-) nuclear localization, seems to be a pathway of minor relevance, since it could not be blocked by tyrosine kinase receptor inhibition. Tyrosine kinase-coupled receptor occupation in general is not protective as demonstrated by the lack of effect with VEGF-infusion.

L12 ANSWER 3 OF 7 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1999:645432 CAPLUS
DOCUMENT NUMBER: 132:31013
TITLE: Angiogenic therapy of acute myocardial infarction by intramyocardial injection of basic fibroblast growth factor
AUTHOR(S): Nagamine, Hiroshi
CORPORATE SOURCE: Department of Surgery (I), School of Medicine, Kanazawa University, Kanazawa, 920-8640, Japan
SOURCE: Kanazawa Daigaku Juzen Igakkai Zasshi (1999), 108(3), 336-348
CODEN: JUZIAG; ISSN: 0022-7226
PUBLISHER: Juzen Igakkai
DOCUMENT TYPE: Journal
LANGUAGE: Japanese

AB Angiogenic growth factors play an important role in the development of coronary collateral circulation. Therapeutic angiogenesis using exogenous growth factors may be useful in patients with severe coronary artery disease in whom complete revascularization is difficult. The present study was performed to examine the effect of one-time intramyocardial injection of basic fibroblast growth factor (bFGF) on myocardial blood flow, vascular d., and ventricular morphol. and function in a canine model of acute myocardial infarction. In 15 anesthetized dogs, myocardial infarction was induced by ligation of the left anterior descending coronary artery (LAD) distal to its first diagonal branch. In 6 of the dogs, **human recombinant** bFGF 100 μg was injected at 8 sites of the **ischemic** area of the left ventricular wall between the LAD and left circumflex coronary artery, while the 9 other dogs served as controls. Myocardial blood flow was determined using the colored microsphere technique both before and immediately after coronary ligation and again 3,7,14, and 28 days after treatment. Myocardial blood flow was expressed as a percentages of the normal flow. Cardiac function was evaluated by repeated echocardiog. measurement. Morphometric anal. was performed in excised hearts by assessing the infarct expansion index and ratio of thinning of infarcted to non-infarcted areas. Angiogenesis was assessed by immunohistochem. stain with anti-von Willebrand factor antibodies. Treatment with bFGF increased endocardial collateral flow in the border zone and the disparity between bFGF-treated and control dogs was significant on day 3 (74.5% and 40.8% in the bFGF and control groups, resp.), with a significant difference on day 7 as well (71.5% and 38.8% in the bFGF and control groups, resp.). Treatment with bFGF increased the epicardial collateral flow in the infarcted zone and the disparity between bFGF-treated and control dogs was significant on day 7 (67.4% and 35.7% in the bFGF and control groups, resp.). Histopathol. examination of the hearts 4 wk after ligation revealed that treatment with bFGF significantly increased the number of capillaries (39.7 and 22.7 per 200 + field in the bFGF and control groups, resp.) and arterioles (4.50 and 2.33 per 200 + field in the bFGF and control groups, resp.) in the border zone. It improved the left ventricular ejection fraction on day 7 after infarction (53.5% and 37.3% in the bFGF and control groups, resp.) and reduced the thinning ratio (43.5% and 26.4% in the bFGF and control groups, resp.). One-time intramyocardial administration of bFGF enhanced collateral vascular development, significantly increased myocardial blood flow and consequently improved global ventricular function. These results suggest that this method has potential as a new therapeutic approach in the treatment of myocardial infarctions.

L18 ANSWER 14 OF 21 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1998:24874 CAPLUS
DOCUMENT NUMBER: 128:106303
TITLE: Adenovirus-mediated expression of the secreted form of basic fibroblast growth factor (**FGF**-2) induces cellular proliferation and angiogenesis in vivo
AUTHOR(S): Ueno, Hikaru; Li, Jian-Jun; Masuda, Satoko; Qi, Zhe; Yamamoto, Hiroaki; Takeshita, Akira
CORPORATE SOURCE: Molecular Cardiology Unit, Department of Cardiology, Kyushu University School of Medicine, Fukuoka, 812-82, Japan
SOURCE: Arteriosclerosis, Thrombosis, and Vascular Biology (1997), 17(11), 2453-2460
CODEN: ATVBFA; ISSN: 1079-5642
PUBLISHER: American Heart Association
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Blood supply through collateral arteries is of critical importance in occlusive arterial diseases such as **coronary** atherosclerosis. Induction of angiogenic growth factor within either the narrowing arteries or jeopardized myocardium may promote angiogenesis in vivo, leading to salvage of **ischemic** myocardium. We constructed a replication-defective adenovirus (AdCAsFGF-2) coding for **human** basic fibroblast growth factor (**FGF**)-2 that is modified, so that its secretion will be facilitated, by tagging a signal sequence derived from **FGF-4**. A large quantity of **FGF**-2 was detected in both the cell lysate and culture medium of COS cells infected with AdCAsFGF-2, indicating that **FGF**-2 was secreted at least partly from the infected cells. The conditioned medium from the infected COS cells stimulated DNA synthesis in and induced cellular proliferation of arterial smooth muscle cells. These effects were eliminated by adenovirus-mediated overexpression of a dominant-neg. truncated **FGF**-receptor type 1. Implantation of a gel of basement membrane proteins containing fibroblasts infected with AdCAsFGF-2 into the ventral s.c. space of mice induced extensive cellular proliferation and the formation of functional arterioles. Cells surrounding the vessels were pos. immunostained with antibodies recognizing either smooth muscle-specific α-actin or factor VIII antigen as a marker for endothelium. These results suggest that AdCAsFGF-2 may be useful for delivering functional **FGF**-2 into tissues and may lead to therapeutic angiogenesis in vivo.

L18 ANSWER 9 OF 21 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2003:664593 CAPLUS
DOCUMENT NUMBER: 140:139562
TITLE: Gene therapy for **coronary** artery disease: preclinical and initial clinical results with intracoronary administration of Ad5FGF-**4**
AUTHOR(S): Watkins, M. W.; Rubanyi, G. M.
CORPORATE SOURCE: Cardiology Unit, University of Vermont College of Medicine, Burlington, VT, 05401, USA
SOURCE: Ernst Schering Research Foundation Workshop (2003), 43(Human Gene Therapy: Current Opportunities and Future Trends), 61-78
CODEN: ESRWEL; ISSN: 0947-6075
PUBLISHER: Springer-Verlag
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AB A review which discusses the preclin. animal data and initial clin. in the largest population of patients to date who have undergone intracoronary administration of gene therapy for myocardial ischemia, specifically with replication-deficient adenovirus containing a **human** fibroblast growth factor gene.
REFERENCE COUNT: 16 THERE ARE 16 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L18 ANSWER 10 OF 21 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2003:610064 CAPLUS
DOCUMENT NUMBER: 139:160389
TITLE: Techniques and compositions for treating cardiovascular disease by in vivo gene delivery of angiogenic peptides and proteins
INVENTOR(S): Hammond, H. Kirk; Dillmann, Wolfgang; Giordano, Frank J.
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 51 pp., Cont.-in-part of U.S. Ser. No. 609,080, abandoned.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 12
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2003148968	A1	20030807	US 2001-847936	20010503
US 5792453	A	19980811	US 1995-485472	19950607
US 6100242	A	20000808	US 1997-722271	19971229
US 6174871	B1	20010116	US 1998-132167	19980810
WO 9940945	A2	19990819	WO 1999-US2702	19990209
WO 9940945	A3	19990930		
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
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AU 9947541	A1	19991125	AU 1999-47541	19990910
WO 2001034208	A1	20010517	WO 2000-US30345	20001103
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ZA 2002003303 A 20030526 ZA 2002-3303 20020425

WO 2002089856 A1 20021114 WO 2002-US13990 20020503

WO 2002089856 C1 20040401

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

US 2004132190 A1 20040708 US 2003-741907 20031219

PRIORITY APPLN. INFO.:

US 1995-396207	B2	19950228
US 1995-485472	A2	19950607
US 1997-852779	B1	19970506
US 1997-722271	A2	19971229
US 1998-21773	B2	19980211
US 1998-68102	B2	19980430
US 1998-132167	A1	19980810
WO 1999-US2702	A2	19990209
US 1999-435156	B2	19991105
US 2000-609080	B2	20000630
WO 2000-US30345	A2	20001103
US 1995-481122	B2	19950607
AU 1996-50287	A3	19960227
WO 1996-US2631	W	19960227
US 1996-660387	B1	19960607
US 1998-98174	B1	19980616
US 2000-664127	A3	20000918
US 2001-847936	A	20010503

AB Methods are provided for treating patients with cardiovascular disease, including heart disease and peripheral vascular disease. The preferred methods of the present invention involve in vivo delivery of genes, encoding angiogenic proteins or peptides, to the myocardium or to peripheral **ischemic** tissue, by introduction of a vector containing the gene into a blood vessel supplying the heart or into a peripheral **ischemic** tissue. A kit comprising a gene therapy composition, a device for introducing the composition into a blood vessel or tissue in vivo, and a vasoactive agent is also claimed.